

Global 'epidemic' of gullet cancer seems to have started in UK in 1950s

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The global "epidemic" of one type of gullet cancer (adenocarcinoma) seems to have started in the UK during the 1950s, sparked by some as yet unknown, but common, factor, suggests research published online in *Gut*.

There are two distinct types of gullet (oesophageal) cancer—squamous and [adenocarcinoma](#), the latter typically affecting the lower third of the oesophagus.

It was first realised that diagnoses of adenocarcinoma were increasing rapidly in several regions of the world in the mid-1980s. But the reasons behind this dramatic increase still remain something of an enigma.

In a bid to identify any discernible patterns, the authors looked at data from 16 population based cancer registries in eight countries, going back between 29 and 54 years.

The eight countries comprised Australia, Denmark, England, Finland, Norway, Scotland, Sweden and the US.

They calculated the rates of the disease that would be expected to be diagnosed, according to the age of the population and looked at the timelines to see if there were particular flash points at which diagnoses suddenly increased.

They found that the incidence of oesophageal adenocarcinoma continued

to increase rapidly in all 16 registries, and there is little evidence that this trend is beginning to plateau.

Men continue to be between three and nine times as likely to develop the disease as women—a pattern that has remained consistent over time.

Although experienced at different periods, with a gap of around 30 years, in around half the registers, a low and seemingly stable rate of new diagnoses shifted abruptly to a rapidly and consistently linear increase.

In the remainder of the registries, the rate had gone beyond a low and stable phase, and was already rising when registration started.

The timeline analysis indicated that the first discernible surge probably occurred around 1960 in Scotland and England, with a similar surge in the US starting in the mid-1970s, and another beginning in 1991 in Sweden, the last country among those studied, to experience this.

The authors suggest that as the analysis was restricted to countries and regions with reliable and longstanding data, it seems unlikely that more than a fraction of these patterns could be attributable to mistakes in correctly identifying the cases.

And as there has been little change in the proportion of patients with localised disease or death rates, changes in diagnostic accuracy are unlikely to explain the trends either.

Obesity, which is linked to acid reflux—a well known cause of the disease—has been rising rapidly too, but the pattern does not fully match that of oesophageal adenocarcinoma, say the authors, nor does it explain the gender difference seen in this type of cancer.

Smoking, an increase in acid reflux, and a fall in *Helicobacter pylori*

infections have all probably played a part in rising incidence, say the authors. But they add: "They are unlikely to explain either the abrupt change or the astounding rate of increase."

And they conclude: "Therefore, it seems reasonable to hypothesise that the effects of a strong, highly prevalent and yet unidentified causal factor—first introduced in the UK in the middle of the 20th century—have been superimposed on the effects of known risk factors."

More information: A global assessment of the oesophageal adenocarcinoma epidemic, Online First, [doi:10.1136/gutjnl-2012-302412](https://doi.org/10.1136/gutjnl-2012-302412)

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